File 155:MEDLINE(R) 1951-2006/Mar 07
(c) format only 2006 Dialog
File 55:Biosis Previews(R) 1993-2006/Feb W4
(c) 2006 BIOSIS
File 34:SciSearch(R) Cited Ref Sci 1990-2006/Feb W4
(c) 2006 Inst for Sci Info
File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
(c) 1998 Inst for Sci Info
File 340:CLAIMS(R)/US Patent 1950-06/Mar 07
(c) 2006 IFI/CLAIMS(R)
***File 340**: IPCR/8 classification codes now searchable in 2006 records.
For important information about IC=index changes, see HELP NEWSIPCR.

Set	Items	Description
? s 20q13.2		
S1	18	20Q13.2

? rd

>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
S2 17 RD (unique items)
? t s2/3,k,ab/1-17

2/3,K,AB/1 (Item 1 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015496260 BIOSIS NO.: 200510190760
Amplification and overexpression of Aurora kinase A (AURKA) in immortalized human ovarian epithelial (HOSE) cells
AUTHOR: Chung C M; Man C; Jin Y; Jin C; Guan X Y; Wang Q; Wan T S K; Cheung A L M; Tsao S W (Reprint)
AUTHOR ADDRESS: Univ Hong Kong, Fac Med, Dept Anat, Canc Biol Lab, Room L1-53 Lab Block,21 Sassoon Rd, Hong Kong, Hong Kong, Peoples R China** Peoples R China
JOURNAL: Molecular Carcinogenesis 43 (3): p165-174 JUL 2005 2005
ISSN: 0899-1987
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Immortalization is an early and essential step of human carcinogenesis. Amplification of chromosome 20q has been shown to be a common event in immortalized cells and cancers. We have previously reported that gain and amplification of chromosome 20q is a non-random and common event in immortalized human ovarian surface epithelial (HOSE) cells. The chromosome 20q harbors genes including TGIF2 (20q11.2-q12), AIB1 (20q12), PTPN1 (20q13.1), ZNF217 (20q13.2), and AURKA (20q13.2-q13.3), which were previously reported to be amplified and overexpressed in ovarian cancers. Some of these genes may be involved in immortalization of HOSE cells and represent crucial premalignant changes in ovarian surface epithelium. Investigation of the involvement of these genes was examined in four pairs of pre-crisis (preimmortalized) and post-crisis (immortalized) HOSE cells. Overexpression of AURKA (Aurora kinase A), also known as BTAK and STK15, by both real time-quantitative polymerase chain reaction (RT-QPCR) and Western blotting was detected in all the four immortalized HOSE cells examined while overexpression of

AIB1 and ZNF217 was observed in two of four immortalized HOSE cells examined. Overexpression of TGIF2 and PTPN1 was not significant in our immortalized HOSE cell systems. The degree of overexpression of AURKA was shown to be closely associated with the amplification of chromosome 20q in immortalized HOSE cells. Fluorescence in situ hybridization (FISH) with labeled Pi artificial clone (PAC) confirmed the amplification of the chromosomal region (20q13.2-13.3) where AURKA resides. DNA amplification of AURKA was also confirmed using semi-quantitative PCR. Our study showed that amplification and overexpression of AURKA is a common and significant event during immortalization of HOSE cells and may represent an important premalignant change in ovarian carcinogenesis. (c) 2005 Wiley-Liss, Inc.
DESCRIPTORS:
...ORGANISMS: PARTS ETC: 20q11.2, 20q13.1, 20q12, **20q13.2**

2/3,K,AB/2 (Item 2 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015451705 BIOSIS NO.: 200510146205
High resolution analysis of DNA copy number changes within the 20q amplicon in adenocarcinomas of different origin
AUTHOR: Carvalho B (Reprint); Coffa J; Buffart T E; Hermsen M A J A; Anders N; Mongera S; Postma C; Schouten J P; Ylstra B; Meijer G A
AUTHOR E-MAIL ADDRESS: b.carvalho@vumc.nl
JOURNAL: Cellular Oncology 27 (2): p99 05 2005
CONFERENCE/MEETING: Meeting of the International-Society-for-Cellular-Oncology (ISCO 2005) Belfast, NORTH IRELAND April 05 -08, 2005; 20050405
SPONSOR: Int Soc Cellular Oncol
ISSN: 1570-5870
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

DESCRIPTORS:
...ORGANISMS: PARTS ETC: 20q11.2, **20q13.2** , long arm

2/3,K,AB/3 (Item 3 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015395626 BIOSIS NO.: 200510090126
Association between beta-adrenergic receptor polymorphisms and their G-protein-coupled receptors with body mass index and obesity in women: a report from the NHLBI-sponsored WISE study
AUTHOR: Terra S G; McGorray S P; McNamara D M; McNamara D M; Cavallari L H; Walker J R; Wallace M R; Johnson B D; Merz C N Bairey; Sopko G; Pepine C J; Johnson J A (Reprint)
AUTHOR ADDRESS: Univ Florida, Coll Pharm, Dept Pharm Practice, POB 100486, Gainesville, FL 32610 USA**USA
AUTHOR E-MAIL ADDRESS: johnson@cop.ufl.edu
JOURNAL: International Journal of Obesity 29 (7): p746-754 JUL 05 2005
ISSN: 0307-0565
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: OBJECTIVES: The beta-adrenergic receptor (beta AR) genes are candidate genes for obesity because of their roles in energy homeostasis and promotion of lipolysis in human adipose tissue. Objective is to determine the association between obesity and polymorphisms in genes of the beta(1)AR (ADRB1), beta(2)AR (ADRB2), beta(3)AR (ADRB3), Gs protein alpha (GNAS1), to which all three beta-receptors couple and the G protein beta 3 subunit (GNB3), to which beta(3)ARs couple.DESIGN: A case-control genetic association study.SUBJECTS: A total of 643 black or white women enrolled in Women's Ischemia Syndrome Evaluation (WISE) study.MEASUREMENTS: Genotypes were determined by PCR with single primer extension. Associations between genotype and body mass index (BMI), waist-to-hip ratio (WHR), waist circumference, and obesity were made.RESULTS: Polymorphisms in the three bAR genes, GNAS1, and GNB3 were not associated with BMI, WHR, waist circumference, or obesity. Linear and logistic regression analyses found no contribution of either genotype or haplotype with anthropometric measurements or obesity.CONCLUSIONS: Our study suggests that among American women with suspected coronary heart disease, polymorphisms in the bARs and their G-protein-coupled receptors do not contribute to increased BMI, WHR, waist circumference, or obesity. Given that 50% of all women die from coronary heart disease, and a higher percentage have heart disease during their lifetime, our results are likely generalizable to many American women.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/4 (Item 4 from file: 55)

0015387120 BIOSIS NO.: 200510081620
High resolution analysis of DNA copy number changes in malignancies with chromosome 20q gains
AUTHOR: Coffa J (Reprint); Hermsen M A J A; Carvalho B; Schouten J P; Meijer G A
AUTHOR ADDRESS: VU Univ, Med Ctr, Dept Pathol, Amsterdam, Netherlands** Netherlands
JOURNAL: Journal of Pathology 204 (Suppl. S): p48A SEP 04 2004
CONFERENCE/MEETING: 186th Meeting of the Pathological-Society-of-Great-Britain-and-Ireland/Dutch-Pathological-Society Amsterdam, NETHERLANDS July 06 -09, 2004; 20040706
SPONSOR: Pathol Soc Great Britain & Ireland
Dutch Pathol Soc
ISSN: 0022-3417
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Citation
LANGUAGE: English

DESCRIPTORS:
...ORGANISMS: PARTS ETC: 20q13.3, 20q11, long arm gain, **20q13.2**

2/3,K,AB/5 (Item 5 from file: 55)

0015365928 BIOSIS NO.: 200510060428
Heredofamilial brain calcinosis syndrome
AUTHOR: Baba Yasuhiko; Broderick Daniel F; Uitti Ryan J; Hutton Michael L; Wszolek Zbigniew K (Reprint)
AUTHOR ADDRESS: Mayo Clin, Coll Med, Dept Neurol, 4500 San Pablo Rd, Jacksonville, FL 32224 USA**USA
AUTHOR E-MAIL ADDRESS: wszolek.zbigniew@mayo.edu
JOURNAL: Mayo Clinic Proceedings 80 (5): p641-651 MAY 05 2005
ISSN: 0025-6196
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Brain calcinosis syndrome (BCS) usually is defined as bilateral calcium accumulation in the brain parenchyma, primarily in the basal ganglia. More than 50 reported clinical conditions have been associated with BCS. We reviewed clinical, radiological, and genetic features of heredofamilial BCS accompanying all conditions associated with calcium accumulation in the brain reported in English between 1962 and 2003 in MEDLINE. The location, extent, and degree of calcification in the brain show diversity not only among the various disorders but also among patients sharing the same condition. The pathogenesis of BCS is uncertain. More complicated mechanisms may be involved when brain calcinosis is present but calcium, phosphorus, and parathyroid hormone metabolism abnormalities are absent. We review conditions associated with heredofamilial BCS in which brain calcinosis is nearly uniformly present because such information may be important to the clinician pursuing an investigative strategy.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: 20q13.3, **20q13.2** ...

2/3,K,AB/6 (Item 6 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015320701 BIOSIS NO.: 200510015201
Use reference bands to accurately estimate ISCN band levels 400, 550, and **850**
AUTHOR: Zabawski James (Reprint); Wiktor Anne; Sikora Matthew; Van Dyke Daniel L
AUTHOR ADDRESS: Henry Ford Hosp, Dept Med Genet, 2799 W Grand Blvd, Detroit, MI 48202 USA**USA
AUTHOR E-MAIL ADDRESS: Jzabaws1@hfhs.org
JOURNAL: Journal of the Association of Genetic Technologists 31 (1): p9-13 05 2005
ISSN: 1523-7834
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In their 2002 Guidelines for chromosome analysis of peripheral blood, the American College of Medical Genetics states that "The 550-band stage should be the goal of all constitutional studies...." The College of American Pathologists requires that the average case be analyzed at the 400-band level of resolution for routine work, and that the 550-band level be achieved in appropriate blood samples. The challenge is how to

identify the 400, 550, and 850-band levels confidently and consistently. In this study, our objectives were to develop simple and reliable criteria to estimate band level, and to evaluate our laboratory's performance with respect to those criteria. Using the ISCN(1995) ideogram as a reference, candidate bands were selected for the three band levels: 400, 550 and 850. A pilot and two follow-up studies were conducted and a set of candidate bands were validated against the Vancouver method of evaluating band level so that band level scores were similar using either method. The final set of reference bands were the presence of 9q32 and 20q13.2 for the 400-band level; 5q33.2 and 10q22.2 for the 550-band level; and 3p26.1, 18q22.3 and 20q13.32 for the 850-band level. Cell selection improved after each technologist was provided a composite image of chromosomes with reference bands highlighted. The band level criteria presented here involve no band counting, appear to be objective, can help to improve quality and consistency among technologists, and can ensure compliance with regulatory agencies.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2** , 20q13.32

2/3,K,AB/7 (Item 7 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015263253 BIOSIS NO.: 200500169989
Genomic alterations in primary gastric adenocarcinomas correlate with clinicopathological characteristics and survival
AUTHOR: Weiss Marjan M; Kuipers Ernst J; Postma Cindy; Snijders Antoine M; Pinkel Daniel; Meuwissen Stefan G M; Albertson Donna; Meijer Gerrit A (Reprint)
AUTHOR ADDRESS: Med CtrDept Pathol, VU Univ, POB 7057, NL-1007 MB, Amsterdam, Netherlands**Netherlands
AUTHOR E-MAIL ADDRESS: ga.meijer@vumc.nl
JOURNAL: Cellular Oncology 26 (5-6): p307-317 2004 2004
MEDIUM: print
ISSN: 1570-5870 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Background & aims: Pathogenesis of gastric cancer is driven by an accumulation of genetic changes that to a large extent occur at the chromosomal level. In order to investigate the patterns of chromosomal aberrations in gastric carcinomas, we performed genome-wide microarray based comparative genomic hybridisation (microarray CGH). With this recently developed technique chromosomal aberrations can be studied with high resolution and sensitivity. Methods: Array CGH was applied to a series of 35 gastric adenocarcinomas using a genome-wide scanning array with 2275 BAC and P1 clones spotted in triplicate. Each clone contains at least one STS for linkage to the sequence of the human genome. These arrays provide an average resolution of 1.4 Mb across the genome. DNA copy number changes were correlated with clinicopathological tumour characteristics as well as survival. Results: All thirty-five cancers showed chromosomal aberrations and 16 of the 35 tumours showed one or more amplifications. The most frequent aberrations are gains of 8q24.2, 8q24.1, 20q13.12, 20q13.2, 7p11.2, 1q32.3, 8p23.1-p23.3, losses of 5q14.1, 18q22.1, 19p13.12-p13.3, 9p21.3-p24.3, 17p13.1-p13.3, 13q31.1,

16q22.1, 21q21.3, and amplifications of 7q21-q22, and 12q14.1-q21.1. These aberrations were correlated to clinicopathological characteristics and survival. Gain of 1q32.3 was significantly correlated with lymph node status (p = 0.007). Tumours with loss of 18q22.1, as well as tumours with amplifications were associated with poor survival (p = 0.02, both). Conclusions: Microarray CGH has revealed several chromosomal regions that have not been described before in gastric cancer at this frequency and resolution, such as amplification of at 7q21-q22 and 12q14.1-q21.1, as well gains at 1q32.3, 7p11.2, and losses at 13q13.1. Interestingly, gain of 1q32.3 and loss of 18q22.1 are associated with a bad prognosis indicating that these regions could harbour gene(s) that may determine aggressive turnout behaviour and poor clinical outcome.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: 20q13.12, **20q13.2** ...

2/3,K,AB/8 (Item 8 from file: 55)

0015197695 BIOSIS NO.: 200500104760
Chromosomal alterations in colorectal carcinomas detected by fluorescence in situ hybridization and comparative genomic hybridization and their **relationship with adenoma-carcinoma sequence: Review analysis of experience at a single Japanese cancer unit**
AUTHOR: Nanashima Atsushi; Yasutake Toru (Reprint); Sawai Terumitsu; Hidaka Shigekazu; Tsuji Takashi; Tagawa Yutaka; Nakagoe Tohru; Tomita Masao; Nagayasu Takeshi
AUTHOR ADDRESS: Grad Sch Biomed SciDiv Surg Oncol, Nagasaki Univ, 1-7-1 Sakamoto, Nagasaki, 8528501, Japan**Japan
AUTHOR E-MAIL ADDRESS: toru@net.nagsaki-u.ac.jp
JOURNAL: Acta Medica Nagasakiensia 49 (1-2): p25-32 June 2004 2004
MEDIUM: print
ISSN: 0001-6055
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: To clarify the relationship with development of colorectal cancer, we investigated chromosomal aberrations in 715 specimens of the colorectal neoplasm by cytogenetic analysis. A gain of chromosome 17 was observed in the transitional epithelium around non-polypoid carcinomas, although the normal epithelium exhibited diploidy. Most tubular adenomas were diploid, however, loss of chromosome 11 and gain of chromosome 17 were increased in adenomas in association with an increased villous component. DNA aneuploidy, aneusomy and p53 deletion were predominantly observed in carcinomas, even in early cancers. Alterations of chromosomes 11 and 18 reflected different tumor morphologies in the early carcinomas. Gains of chromosomes 11, 17 and 18, and deletion of chromosomes 11 and 17p and p53 became more frequent following an increase in the depth of invasion. Aneusomy of chromosome 11 was a risk factor for patient survival after operation. Gains of chromosome 20 and 20q13.2 were associated with liver metastasis. Aneusomy and translocations of chromosome 17 and the p53 locus were predominantly observed in patients with multiple cancers and hereditary non-polyposis colorectal cancer. Our results indicate that in the process of development of colorectal carcinomas, specific chromosomal aberrations might be related to each

step of development, or an alternative pathway of de novo carcinogenesis.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/9 (Item 9 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015189714 BIOSIS NO.: 200500095627
Overexpression and amplification of STK15 in human gliomas
AUTHOR: Klein Alexandra; Reichardt Wilfried; Jung Volker; Zang Klaus D; Meese Eckart; Urbschat Steffi (Reprint)
AUTHOR ADDRESS: Inst Human Genet, Univ Saarlandes, Bldg 60, D-66421, Homburg, Germany**Germany
AUTHOR E-MAIL ADDRESS: hgsmur@uniklinik-saarland.de
JOURNAL: International Journal of Oncology 25 (6): p1789-1794 December 2004 2004
MEDIUM: print
ISSN: 1019-6439 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The serine/threonine kinase 15 (STK15) at chromosome 20q13.2 is frequently shown to be amplified and overexpressed in several human cancers. STK15 has been reported to act as a cell cycle regulator and its overexpression induces centrosome amplification and aneuploidy. Recently we showed that STK15 even plays a role in human malignant brain tumours and we described an amplification of the gene in 31% of the investigated gliomas. In this study we scrutinized the correlation of increased STK15 on DNA and mRNA levels in gliomas of different histological grades. Southern blotting confirmed the amplification frequency of the STK15 gene, which had been previously detected by comparative PCR. In total, DNA gains were found in 26% of the investigated gliomas. Interestingly, we detected overexpression of STK15 mRNA in 60% of the analyzed brain tumours. The elevated expression does not strongly correlate with gains on DNA level, but all cases with an amplification of the STK15 gene display overexpression. Gains of the STK15 gene seem to occur irrespectively of the histological grades of the tumours, so that STK15 probably is not a progression associated factor. Amplification and overexpression of the kinase rather represent a primary alteration in human gliomas, which could play an important role as an early event in all glioma subtypes.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/10 (Item 10 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015169250 BIOSIS NO.: 200500076315
Physical and functional interactions between the Wwox tumor suppressor protein and the AP-2gamma transcription factor

AUTHOR: Aqeilan Rami I; Palamarchuk Alexey; Weigel Ronald J; Herrero Juan J ; Pekarsky Yuri; Croce Carlo M (Reprint)
AUTHOR ADDRESS: Ctr Comprehens CancDept Mol Virol Immunol and Med GenetHuman Canc Genet Program, Ohio State Univ, 400 W 12th St,Weisman 435, Columbus, OH, 43210, USA**USA
AUTHOR E-MAIL ADDRESS: Carlo.Croce@mail.tju.edu
JOURNAL: Cancer Research 64 (22): p8256-8261 November 15, 2004 2004
MEDIUM: print
ISSN: 0008-5472 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The WWOX gene encodes a tumor suppressor WW domain-containing protein, Wwox. Alterations of WWOX have been demonstrated in multiple types of cancer, and introduction of Wwox into Wwox-negative tumor cells has resulted in tumor suppression and apoptosis. The Wwox protein contains two WW domains that typically bind proline-rich motifs and mediate protein-protein interactions. Recently, we have described functional cross-talk between the Wwox protein and the p53 homologue, p73. To further explore the biological function of Wwox, we investigated other interacting candidates. In this report, we demonstrate a physical and functional association between AP-2gamma transcription factor and the Wwox protein. AP-2gamma at 20q13.2 encodes a transcription factor and is frequently amplified in breast carcinoma. We show that Wwox binds to the PPPY motif of AP-2gamma via its first WW domain. Alterations of tyrosine 33 in the first WW domain of Wwox or the proline-rich motif in AP-2gamma dramatically reduce this interaction. In addition, our results demonstrate that Wwox expression triggers redistribution of nuclear AP-2gamma to the cytoplasm, hence suppressing its transactivating function. Our results suggest that Wwox tumor suppressor protein inhibits AP-2gamma oncogenic activity by sequestering it in the cytoplasm.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/11 (Item 11 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015146743 BIOSIS NO.: 200500053808
The candidate oncogene ZNF217 is frequently amplified in colon cancer
AUTHOR: Rooney Patrick H; Boonsong Attasit; McFadyen Morag C E; McLeod Howard L; Cassidy James; Curran Stephanie; Murray Graeme I (Reprint)
AUTHOR ADDRESS: Dept Pathol, Univ Aberdeen, Foresterhill, Aberdeen, AB25 2ZD, UK**UK
AUTHOR E-MAIL ADDRESS: g.i.murray@abdn.ac.uk
JOURNAL: Journal of Pathology 204 (3): p282-288 November 2004 2004
MEDIUM: print
ISSN: 0022-3417 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: In this study we have defined the changes in gene copy number of the candidate oncogene ZNF217 during colon cancer development and progression. This gene is mapped to chromosome 20q and lies within

20q13.2, a region which we have previously shown to be highly amplified in colorectal cancer by comparative genomic hybridization. The gene copy number of ZNF217 was assessed in 100 colon carcinomas (19 Dukes' A, 42 Dukes' B and 39 Dukes' Q, 13 colonic adenomas and 10 normal colon samples. DNA extracted from laser microdissected cells was amplified by multiplex real-time PCR at two distinct gene loci - ZNF217 and beta-globin (control gene) - on an AB17700 sequence detection system. Of the 100 colon cancers studied, 61 showed some level of amplification of ZNF217, 15 had loss of ZNF217, while 24 were diploid. All the adenomas except one were diploid. In this study we have found that ZNF217 amplification is a frequent event in colon cancer and that the extent of its amplification varies markedly between tumours (range 3-13 copies). There was a trend toward poorer survival in patients whose cancers had either gain or loss of ZNF217. Copyright Copyright 2004 Pathological Society of Great Britain and Ireland. Published by John Wiley & Sons, Ltd.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/12 (Item 12 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0015123264 BIOSIS NO.: 200500030329
A genome scan for ESRD in black families enriched for nondiabetic nephropathy
AUTHOR: Freedman Barry I (Reprint); Langefeld Carl D; Rich Stephen S; Valis Christopher J; Sale Michele M; Williams Adrienne H; Brown W Mark; Beck Stephanie R; Hicks Pamela J; Bowden Donald W
AUTHOR ADDRESS: Bowman Gray Sch MedNephrol SectDept Internal Med, Wake Forest Univ, Med Ctr Blvd, Winston Salem, NC, 27157, USA**USA
AUTHOR E-MAIL ADDRESS: bfreedma@wfubmc.edu
JOURNAL: Journal of the American Society of Nephrology 15 (10): p 2719-2727, 2713 October 2004 2004
MEDIUM: print
ISSN: 1046-6673
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Nephropathy is a complex disorder, with predisposition influenced by the interplay of both genetic and environmental factors. As part of an effort to map genes that predispose to ESRD, a genome scan was performed in 264 black pedigrees that contained 296 ESRD-affected sibling pairs using multipoint nonparametric linkage analysis methods. The cause of ESRD in index cases was consistent with hypertension-associated ESRD. Nonparametric linkage (NPL) regression provided modest evidence of linkage to 9p21.3 near D9S1121 (logarithm of odds (LOD) = 2.03), 1q25.1 near D1S1589 (LOD = 1.62), and 13q33.3 near D13S796 (LOD = 1.02). Adjusting for the evidence of linkage at the other loci through the NPL regression analysis provided evidence for linkage to 1q25.1, 6p23, and 9p21.3. The NPL regression and ordered subset analyses suggest that the evidence for linkage significantly increased with early onset of ESRD (2q32.1 LOD = 3.89, 13q13.1 LOD = 3.90), increased BMI (8p22 LOD = 3.37, 13q33.3 LOD = 5.20, 18p11.3 LOD = 2.38), early onset of hypertension (14q21.1 LOD = 3.19, 20q13.2 LOD = 2.32), and late onset of hypertension

(4q13.1 LOD = 3.44, 5p15.33 LOD = 2.82). Multipoint single-locus linkage analysis provided modest evidence of linkage to nondiabetic ESRD on 9p21.3, 1q25.1 (in the region of the podocin gene), and 13q33.3. NPL regression and ordered subset analyses also identified loci on 13q13.1 and 13q33.3 as contributing to early-onset ESRD and ESRD in the presence of increased BMI, respectively. These regions should receive priority in the search for loci that contribute susceptibility to nondiabetic nephropathy.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/13 (Item 13 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0014697841 BIOSIS NO.: 200400068598
Amplicon in the 20q13 region of human chromosome 20 and uses thereof
AUTHOR: Albertson Donna G (Reprint); Pinkel Daniel; Collins Colin Conrad; Gray Joe W
AUTHOR ADDRESS: San Rafael, CA, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1277 (3): Dec. 16, 2003 2003
MEDIUM: e-file
PATENT NUMBER: US 6664057 PATENT DATE GRANTED: December 16, 2003 20031216
PATENT CLASSIFICATION: 435-6 PATENT ASSIGNEE: The Regents of the University of California PATENT COUNTRY: USA
ISSN: 0098-1133 (ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This invention pertains to the field of cancer genetics and cytogenetics. In particular, this invention pertains to the identification of a novel amplicon on human chromosome 20 which is associated with cancer. More particularly this invention pertains to the identification of a novel "amplicon" or genomic nucleic acid in a region of amplification at about 20q13.2 which has been associated with a variety of cancers, particularly breast cancer. The novel amplicon of the invention can be used as a probe specific for this region of 20q13.2 as well as for the diagnosis and prognosis of various cancers. Also provided are kits for screening for the presence and copy number of the novel amplicon of the invention in a sample containing human nucleic acid.

DESCRIPTORS:
...ORGANISMS: PARTS ETC: **20q13.2**

2/3,K,AB/14 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

13493732 Genuine Article#: 888BZ Number of References: 29
Title: Association of 20q13.2 copy number changes with the advanced stage of ovarian cancer - tissue microarray analysis (ABSTRACT AVAILABLE)
Author(s): Dimova I (REPRINT) ; Yosifova A; Zaharieva B; Raitcheva S; Doganov N; Toncheva D

Corporate Source: Med Univ Sofia,Dept Med Genet,2 Zdrave Str/Sofia 1431//Bulgaria/ (REPRINT); Med Univ Sofia,Dept Med Genet,Sofia 1431//Bulgaria/; Univ Hosp Maichin Dom Sofia,Gynecopathol Unit,Sofia 1431//Bulgaria/; Univ Hosp Maichin Dom Sofia,Gynecol Clin 2,Sofia 1431//Bulgaria/(dragatoncheva@yahoo.com)
Journal: EUROPEAN JOURNAL OF OBSTETRICS GYNECOLOGY AND REPRODUCTIVE BIOLOGY , 2005, V118, N1 (JAN 10), P81-85
ISSN: 0301-2115 Publication date: 20050110
Publisher: ELSEVIER SCI IRELAND LTD, CUSTOMER RELATIONS MANAGER, BAY 15, SHANNON INDUSTRIAL ESTATE CO, CLARE, IRELAND
Language: English Document Type: ARTICLE
Abstract: Overrepresentations in 20q have been reported in a number of ovarian cancers by comparative genomic hybridization. In order to study the relation of the increased copy number of 20q13.2 with tumor phenotype in ovarian cancer, we applied FISH on a tissue microarray. The TMA technology enables us to analyze a large number of different malignancy, histology, stage and grade tumors. Overall, the frequency of 20q13.2 alterations in epithelial ovarian cancer was 25.50% (10.74% gains and 14.76% amplifications). There was not statistically significant difference between the frequencies of 20q13.2 copy number changes in different grade tumors. The frequency of gains and amplifications increased significantly from stage I to stage II to stage III tumors. Our results showed strong association between increases 20q13.2 copies and advanced tumor stage. We concluded that genetic alterations in 20q13.2 may be of prognostic significance for stage progression of the ovarian cancer.

2/3,K,AB/15 (Item 2 from file: 34)

12500056 Genuine Article#: 774HJ Number of References: 17
Title: Clinical significance of the overexpression of the candidate oncogene CYP24 in esophageal cancer (ABSTRACT AVAILABLE)
Author(s): Mimori K; Tanaka Y; Yoshinaga K; Masuda T; Yamashita K; Okamoto M; Inoue H; Mori M (REPRINT)
Corporate Source: Kyushu Univ,Med Inst Bioregulat, Dept Surg,Tsurumihara 4546/Beppu/Oita 8740838/Japan/ (REPRINT); Kyushu Univ,Med Inst Bioregulat, Dept Surg,Beppu/Oita 8740838/Japan/; Saitama Canc Ctr,Dept Surg,Saitama//Japan/
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Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND
Language: English Document Type: ARTICLE
Abstract: Background: By using array comparative genomic hybridization (CGH), the increased copy number of CYP24 (which encodes vitamin D 24-hydroxylase) at 20q13.2 was previously reported, leading to the identification of CYP24 as a candidate oncogene in breast cancer. CYP24 leads to abrogate growth control mediated by vitamin D.

Materials and methods: We examined CYP24 expression as well as VDR (vitamin D receptor) gene expression in 42 esophageal cancer cases using semi-quantitative RT-PCR assay. We induced CYP24 in seven esophageal cancer cell lines using 25-hydroxyvitamin D-3 [25(OH)D-3] and compared cell growth rate, measured using the 3-(4, 5-dimethylthiazol-2-y)-2, 5-diphenyltetrazolium bromide (MTT) assay system.

Results: The overall survival rate was significantly higher in 25 cases of lower CYP24 expression than 17 cases of higher CYP24 expression ($P < 0.05$); on the other hand, 23 cases of low VDR expression had a poorer prognosis than 19 cases of high VDR expression. Moreover, we disclosed that the inverse correlation between CYP24 and VDR expression is significant in esophageal cancer cases ($P < 0.05$). Furthermore, the cell growth evaluated by MTT assay was greatly increased in CYP24-induced and VDR-diminished cells than non-responding cells by 25(OH)D3 activity ($P < 0.01$).

Conclusions: Overexpression of the candidate oncogene CYP24 is inversely correlated to vitamin D receptor expression, and may play an important role in determination of the malignant potential of esophageal cancer.

2/3,K,AB/16 (Item 3 from file: 34)

11800288 Genuine Article#: 697FJ Number of References: 48

Title: Determination of amplicon boundaries at 20q13.2 in tissue samples of human gastric adenocarcinomas by high-resolution microarray comparative genomic hybridization (ABSTRACT AVAILABLE)

Author(s): Weiss MM; Snijders AM; Kuipers EJ; Ylstra B; Pinkel D; Meuwissen SGM; van Diest PJ; Albertson DG; Meijer GA (REPRINT)
Corporate Source: Vrije Univ Amsterdam,Med Ctr, Dept Pathol,POB 7057/NL-1007 MB Amsterdam//Netherlands/ (REPRINT); Vrije Univ Amsterdam,Med Ctr, Dept Pathol,NL-1007 MB Amsterdam//Netherlands/; Vrije Univ Amsterdam,Med Ctr, Microarray Core Facil,NL-1007 MB Amsterdam//Netherlands/; Erasmus Univ,Med Ctr, Dept Gastroenterol & Hepatol,Rotterdam//Netherlands/; Univ Calif San Francisco,Ctr Comprehens Canc,San Francisco//CA/94143; Vrije Univ Amsterdam,Med Ctr, Dept Gastroenterol,Amsterdam//Netherlands/

Journal: JOURNAL OF PATHOLOGY, 2003, V200, N3 (JUL), P320-326
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Publisher: JOHN WILEY & SONS LTD, THE ATRIUM, SOUTHERN GATE, CHICHESTER PO19 8SQ, W SUSSEX, ENGLAND
Language: English Document Type: ARTICLE

Abstract: Comparative genomic hybridization (CGH) of gastric adenocarcinomas frequently shows gains and amplifications of chromosome 20. However, the underlying genetic lesion is unknown and conventional CGH results do not allow specification of the target region. In order to investigate this chromosomal aberration with a higher resolution and sensitivity, microarray-based CGH was performed with both scanning and high-resolution arrays of chromosome 20 in a series of 27 gastric adenocarcinomas. Locus-specific fragments of genomic DNA from bacterial artificial chromosome (BAC) clones were spotted as microarrays. A scanning array contained a set of 27 BAC clones covering chromosome 20q. A high-resolution array contained 27 overlapping BAC clones at 20q13.2. This high-resolution array was used to narrow down the amplicon at 20q13.2 in tumours showing amplification of this chromosomal region with the scanning array. Positive copy number changes on chromosome 20q were detected in 12 of 27 cases (44%). These changes included gain of the whole arm of chromosome 20q in 8 of 27 (30%) cases, amplification restricted to 20q12.1 in one case, and amplifications restricted to 20q13 in three cases (11%). The three tumours showing amplification restricted to 20q13 were analysed further

using the high-resolution array. In one tumour, the whole contig was amplified at a constant level. One of the other two tumours had a clear proximal breakpoint, while the other tumour had a clear distal breakpoint within the 20q13.2 region. The proximal and the distal breakpoint were approximately 800 kb apart. In the present study, an amplicon at 20q13.2 has been narrowed down to 800 kb which is likely to harbour one or more putative oncogenes relevant to gastric carcinogenesis, for which ZNF217 and CYP24 are good candidates.

2/3,K,AB/17 (Item 4 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

11179593 Genuine Article#: 619JA Number of References: 52
Title: CAS (cellular apoptosis susceptibility) gene expression in ovarian **carcinoma - Correlation with 20q13.2 copy number and cyclin D1, p53, and Rb protein expression** (ABSTRACT AVAILABLE)
Author(s): Peiro G; Diebold J (REPRINT) ; Lohrs U
Corporate Source: Univ Munich,Inst Pathol,Thalkirchner Str 36/D-80337 Munich//Germany/ (REPRINT); Univ Munich,Inst Pathol,D-80337 Munich//Germany/
Journal: AMERICAN JOURNAL OF CLINICAL PATHOLOGY, 2002, V118, N6 (DEC), P 922-929
ISSN: 0002-9173 Publication date: 20021200
Publisher: AMER SOC CLIN PATHOLOGISTS, 2100 W HARRISON ST, CHICAGO, IL 60612 USA
Language: English Document Type: ARTICLE
Abstract: We immunohistochemically analyzed cellular apoptosis susceptibility (CAS) protein expression and compared it with 20q13.2 copy number and the expression of cell cycle-associated proteins retinoblastoma (Rb), cyclin D1, and p53 and prognosis on paraffin-embedded tissue from 69 ovarian carcinomas (OCs).

CAS protein reactivity was present in 100%, Rb in 54%, cyclin D1 in 47%, and p53 in 49%. Significant reciprocal correlation was observed between high levels of CAS and histologic type, FIGO (International Federation of Obstetrics and Gynecology) stage III and grade 3, residual tumor (>2 cm), 20q13.2 (ZNF217 gene) amplification (>4 copies in >20% cells), and high expression of cyclin D1 (all P <.05). No association was found between cyclin D1, p53, or Rb levels with clinicopathologic factors. In univariate analysis, residual tumor, FIGO stage and grade, ZNF217 amplification, and CAS levels predicted outcome (all P <.05). In multivariate analysis, stage, grade, amount of residual tumor, and ZNF217 amplification showed independent prognostic value (all P <.05).

In OC, alteration of CAS and ZNF217 genes, both located at 20q13, is frequent and relevant prognostically. Cyclin D1, Rb, and p53 seem to have a secondary role.
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